adrenergic casualty department, where further delays would take place and possibly further errors by inexperienced junior staff. Unfortunately, the message of the British Heart Foundation report is deeply ambivalent, doubtless reflecting a "dissensus" in the group. The overall result, however, will be to discourage general practitioners from participating fully and exploiting the major benefits that thrombolytic treatment can confer. Rather than "contracting out," as the report suggests, I hope that general practitioners will insist on local schemes to bolster their confidence in the full early management of myocardial infarction.

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Child sexual abuse

SIR,—I am pleased that investigations for sexually transmitted diseases including screening tests for gonorrhoea and HIV infection should be done on sexually abused children.[1]

Over two years five children (two girls, three boys) aged 4-7½ years presented at this teaching hospital, not with a history of sexual abuse but with urethral or vaginal discharge proved to be due to *Neisseria gonorrhoea*. One 5 year old girl subsequently admitted to sexual abuse by a 10 year old boy at school. The boy refused to be investigated by us. The other girl, aged 7½ years, denied sexual abuse and had an intact hymen. Three boys were subsequently found to have contracted the disease through a parent or older member of the family.

Reports on sexual abuse in children in the developing countries are rare,[2] but our experience shows that doctors and, in particular, paediatricians in these countries need to be aware of sexual abuse and that the campaign against HIV infection and other sexually transmitted diseases for at risk subjects should include children who have been sexually abused.

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1 Bamford F, Roberts R. Child sexual abuse—II. *Br Med J* 1989;299:377-82 (5 August).
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Marker for alcohol abuse

SIR,—The prospect of a more reliable marker for alcoholism as described by Mr A Kapur and colleagues is most welcome.[1] Unfortunately, however, their last paragraph states that "the cost of the test compares favourably with that of other standard laboratory investigations." The given method does not specify the reagents closely enough for the costs of consumables to be worked out, but I challenge Mr Kapur and colleagues to produce a result for 22p per specimen (the current cost of consumables for a γ-glutamyltransferase estimation in this department). A full blood count (including mean corpuscular volume) performed by our haematology department represents even better value at 11p for consumables. The isolation and identification of carbohydrate deficient transferrin is patently more labour intensive than either of the above automated methods, and the rather glib dismissal of necessary technician time shows a lack of understanding for the problems of laboratories that will be asked to perform these investigations on a day to day basis, given the current volume of requests for markers of alcohol abuse.

Chemical pathology departments that seek to sell this "fairly simple, sensitive, and inexpensive" technique to their managers and clinicians as an alternative to cheaper current tests (albeit with known limitations) may thus be hoist with their own petard.

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1 Kapur A, Wild G, Milford-Ward A, Triger DR. Carbohydrate deficient transferrin: a marker for alcohol abuse. *Br Med J* 1989;299:427-31 (12 August).

Passive smoking and cardiorespiratory health in Scotland

SIR,—Mr David J Hole and colleagues,[1] when discussing results from their prospective study, state that studies of cotinine in passive smokers suggest that the dose received may be "equivalent to smoking up to three cigarettes a day." To support this misleading statement they cite a solitary study in Japan,[2] in which urinary cotinine concentrations in non-smokers averaged 8% of those in smokers. This contrasts sharply with evidence from Western populations,[3] which indicates that average cotinine concentrations in non-smokers exposed to environmental tobacco smoke are about 0·7% of those in smokers.[4] Blott and Fraumeni speculated that Japanese people might have especially heavy exposure to environmental tobacco smoke.[5] Other studies in Japan[6] (and abstracts presented by S Umemura and colleagues and E Higashi and colleagues, international conference on indoor air quality, Tokyo, 1987) have, however, sustained earlier suspicions[7] that the methodology used in the original study was faulty. When estimating passive exposure relative to that from active smoking nicotine based indices are of dubious value, partly because nicotine in environmental tobacco smoke, unlike that in mainstream smoke, is largely in the vapour phase and need not be absorbed by the lungs.[8] Based on measurements of retained particulate matter, exposure to environmental tobacco smoke averages at about 0·05% of the exposure of a person who smokes 20 cigarettes each day—that is, 0·01 cigarettes a day.

That such minute doses should elicit observable health effects is surprising, and epidemiological studies that report associations with exposure to environmental tobacco smoke have been critically examined for possible bias. One important bias arises because some smokers deny present or past smoking. Mr Hole and colleagues refer to one of my papers,[9] but unfortunately have totally misunderstood how such bias arises. They state that differential rates of misclassification imply that someone in their "double smoking group" has to be "more likely to pretend to be a non-smoker than someone in the single smoking group." This is untrue because it overlooks the fact that smokers tend to cohabit with smokers.

The table shows how differential misclassification can arise, assuming 2% of the index subjects had denied smoking. The higher proportion of smokers (15·6%) in the observed passive smoking group compared with the observed control group (6·8%) would cause substantial bias for an end point strongly related to active smoking. Thus if risk were increased 20 times in smokers, and not by exposure to environmental tobacco smoke, the relative risks observed would be 6·90 for active smoking and 1·74 for passive smoking, not 20 and 1 respectively. Many studies have shown higher rates of denial of smoking than assumed in the table,[10] so this source of bias is evidently important. It can explain the many positive associations reported in the Scottish study,[1] most of which were not statistically significant.

The results for lung cancer from the Scottish study were based on only nine deaths among self reported non-smokers. This contrasts with over 2000 deaths in other published studies. Clearly, the new data contribute little to the overall picture. Evidence on environmental tobacco smoke and heart disease has previously been reviewed and considered inconclusive.[11] Although the Scottish study reported more deaths from heart disease than from lung cancer, it should not materially affect this view.

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Donating drugs to the Third World

SIR,—As director of Intercare, the organisation approved by the BMA Board of Science and Education for promoting the salvaging of suitable medical samples for use in the Third World, I am happy to answer the criticisms expressed by Dr Frances Griffiths.[1]

Differential misclassification caused by 2% of index subjects denying smoking regardless of cohabitee's smoking habits

Exposure group*	Smoking state of index subject	Smoking state of cohabitee	"True" distribution†	Effects of denial	Observed distribution‡	Percentage who have smoked‡
Controls	Non-smoker	Non-smoker	399	−29	428	6·8
Passive smokers	Non-smoker	Smoker	205	+38	243	15·6
Single smokers	Smoker	Non-smoker	1449	−29	1420	
Double smokers	Smoker	Smoker	1907	−38	1869	

*As defined by Hole *et al*. †Data from table I of Hole *et al*. ‡Among the unseen population.